



Dementia - notes on demetia

Neuropsychology (Christ (Deemed To Be University))



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Dementia

Clinical characteristics and etiology and treatment

Neurocognitive Disorders (NCDs)

General Approach to NCD's

- Characterize the patient syndromically
Mild NCD
Major NCD
- Determine etiology of syndrome

Neurocognitive Disorders

Major vs Mild

- 1. Severity**
- 2. Independence**
- 3. Usually a continuum with evolution**
- 4. Etiology**

Neurocognitive Disorders

Domains

Domain

Tasks

Complex attention

Major: diminished, multiple stimuli
Mild: takes longer

Executive abilities

Major: abandon complex activities
Mild: ↑ effort, multi-tasking

Learning/memory

Major: repeat self in conversation
Mild: recent events, occas repeat

Language

Major: aphasia, paraphasias
Mild: ↓ naming, word finding

Visuoconstruction

Major: not driving, ↓ navigation

Visuoperception

Mild: maps, effort

Social cognition

Major: insensitivity social contexts
Mild: subtle personality, ↓ empathy

Mild Neurocognitive Disorder (MCI)

Major Neurocognitive Disorder (Dementia)

Cognition

Independence

1. Cognitive decline
2. Single cognitive domain impaired (usually)
3. Preservation of independence

1. Cognitive decline
2. Significant cognitive impairment in one or more often multiple cognitive domains
3. Loss of independence

Major NCD

Dementia

- A. Cognitive decline (1 or usually 2 cognitive domains)**
 - 1. Report by patient, informant, clinician**
and
 - 2. Deficits on assessment**
- B. Interfere with independence assistance in IADL's**
- C. Not delirium**
- D. Not primarily attributable to another disorder**

Mild NCD

MCI

Rationale

Mild cognitive impairment

Early recognition

Intervention

Clinical trials

Criteria

A. Cognitive decline

- 1. Report by patient, informant, clinician
and**
- 2. Mild cognitive deficits**

B. Not interfere with independence greater effort

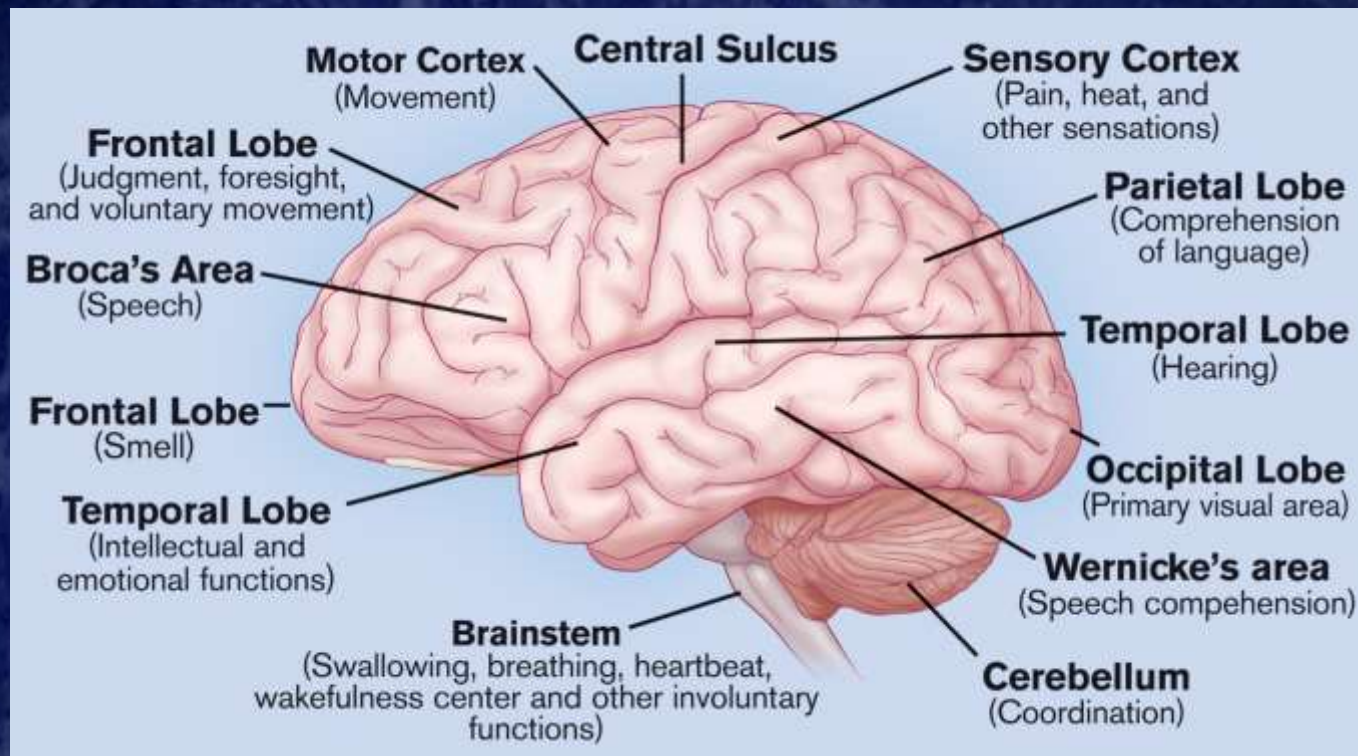
C. Not delirium

D. Not primarily attributable to other disorder

NCD Etiologies- DSM V

- Alzheimer's Disease
- Frontotemporal degen
- Lewy body disorders
- Vascular cognitive impairment
- Traumatic brain injury
- Substance/medications
- HIV/AIDS
- Prion disease
- Parkinson's disease
- Huntington disease
- Other medical issues
- Multiple causes

Brain Structure



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(Wells, & Pasternak, 2019)

Case Study

The patient was referred to our specialty memory clinic at the age of 58 with a 2-year history of repetitiveness, memory loss, and executive function loss. MRI scan revealed mild generalized cortical atrophy resulting. She is white with 2 years of postsecondary education. Retirement at age 48 from employment as a manager in telecommunications company was because family finances allowed and not because of cognitive challenges with work. Progressive cognitive decline was evident by the report of deficits in instrumental activities of daily living performance over the past 9 months before her initial consultation in the memory clinic. Word finding and literacy skills were noted to have deteriorated in the preceding 6 months according to her spouse. Examples of functional losses were being slower in processing and carrying out instructions, not knowing how to turn off the stove, and becoming unable to assist in boat docking which was the couple's pastime. She stopped driving a motor vehicle about 6 months before her memory clinic consultation. Her past medical history was relevant for hypercholesterolemia and vitamin D deficiency. She had no surgical history. She had no history of smoking, alcohol, or other drug misuse. Laboratory screening was normal. There was no first-degree family history of presenile dementia.

(Wells, & Pasternak, 2019)

Alzheimer's Disease- DSM V Criteria

A. Meets the criteria for major or mild neurocognitive disorder

B. Insidious onset and gradual progression of impairment in one or more cognitive domains (to be classified as major at least two domains must be impaired)

C. Criteria for probable or possible Alzheimer's disease based on:

Alzheimer as Major Neurocognitive Disorder:

If causes of genetic mutation from family history or genetic testing

Following three are present:

1. Decline in memory and learning in at least one of the cognitive domain (detailed history or serial neuropsychological testing)
2. Steadily progressive, gradual decline in cognition, without extended plateaus.
3. No evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease, or another neurological, mental, or systemic disease or condition likely contributing to cognitive decline).

Alzheimer's Disease- DSM V Criteria

Alzheimer as Mild Neurocognitive Disorder:

Alzheimer will be diagnosed as probable disease if no evidence of genetic mutation from either genetic testing or family history

Alzheimer will be diagnosed as possible disease if no evidence of genetic mutation from either genetic testing or family history with three of the following conditions being present:

1. Clear evidence of decline in memory and learning.
2. Steadily progressive, gradual decline in cognition, without extended plateaus.
3. No evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease, or another neurological or systemic disease or condition likely contributing to cognitive decline).

D. Rule out disturbances due to cerebrovascular disease, another neurodegenerative disease, the effects of a substance, or another mental, neurological, or systemic disorder

Alzheimer's Disease

Associated Features:

Changes in:

(a) Mood: Features of depression (psychomotor retardation, apathy, crying, poor appetite, disturbed sleep, and expressions of unhappiness) ranging from 10% to 50%. Some 3.5% of individuals may also develop elation of mood, disinhibition, and hypomania.

(b) Psychosis: Some form of delusions such as paranoid delusions (20 %- 70%) or visual hallucinations (20%-28%). In few cases Capgras syndrome may occur.

(c) Personality: Family members have described the loss of personality as a '**living bereavement**'—the body remains, but the person once known has gone. Some of the personality changes are- loss of awareness resulting into anxiety, fearfulness and normal responsiveness to the environment resulting into apathy or flattening of affect. Withdrawal from challenging situations or catastrophic reaction to it

(d) Behavioral: Aggression part of psychosis. Wandering due to topographical confusion, altered sleep-wake cycle as a result sundowning . Excessive or inappropriate vocalizations (grunting and screaming) occur in the late stages.

Causes are attributed to loss of serotonergic and noradrenergic markers accompanies cholinergic loss

(Deardorff, & Grossberg, 2019; Li et al., 2014)

Epidemiology

Alzheimer's is the most common cause of dementia, accounting for an estimated 60% to 80% of cases worldwide.

Alzheimer's disease, a degenerative brain disorder, has been steadily on the rise in India, with a prevalence rate of 7.4 percent among adults above 60 years.

(Lee et al., 2023)

Course And Prognosis

- In the early stages of Alzheimer's disease, the clinical features are modified by the patient's premorbid personality, and their traits tend to be exaggerated. In the middle and later stages of the illness, the cognitive impairments increasingly predominate, together with the neurological and behavioural features.
- Incidental physical illness (e.g., heart and blood circulation problems, stroke, and diabetes) may cause a superimposed delirium, resulting in a sudden deterioration in cognitive function.
- Median survival from diagnosis is 5– 7 years, and is slightly less in men than in women (poor prognosis).
- Shorter survival is also associated with an older age of onset, and a rapid rate of cognitive decline.

(Beam et al., 2020)

Causes

Researchers trying to understand the cause of Alzheimer's disease are focused on the role of two proteins:

- **Plaques.** **Beta-amyloid** is a fragment of a larger protein. When these fragments clump together, they appear to have a toxic effect on neurons and to disrupt communication between brain cells. These clumps form larger deposits called **amyloid plaques**.

- **Tangles.** **Tau proteins** play a part in a brain cell's internal support and transport system to carry nutrients and other essential materials. In Alzheimer's disease, tau proteins change shape and organize into structures called **neurofibrillary tangles**. The tangles disrupt the transport system and cause damage to cells.

Causes

The cholinergic hypothesis

Plaques and tangles first occur in the hippocampus. Some areas of the brain are relatively preserved—the occipital lobe and cerebellum is affected relatively late. The cognitive impairment of AD was due to a disorder predominantly affecting cholinergic neurones.

The amyloid cascade hypothesis

the formation of β -amyloid as the initiating, or at least early event, leading to all the other changes observed including tau aggregation and phosphorylation, neuronal loss, cholinergic deficits, and clinical symptoms.

The presenilin genes

Mutations in *presenilin-1* (*PS-1*) and *presenilin-2* (*PS-2*), two very similar genes on chromosome 14 and chromosome 1 respectively, also cause early onset autosomal dominant AD.

Causes

Molecular genetics

Mutations in three genes have been found to cause early onset familial AD, which is inherited:

- 1) Mutations in the **APP gene (Amyloid-beta precursor protein on chromosome 21)** are the least common
- 2) Mutations in **PS-1 (on chromosome 14)** are somewhat more frequent, although are still a rare cause of AD.
- 3) Mutations in **PS-2 (on chromosome 1)** appear to be largely restricted to an ethnic German people residing in the United States

Mutations in other genes (*tau* and *progranulin*) which is caused for *frontotemporal and related disorders*.

Risk Factors

Individuals with **Down's syndrome** are at extremely high risk of AD, with neuropathological evidence being present in virtually all individuals living to middle age, probably because of trisomy APP (on chromosome 21).

Family history of dementia is the largest single risk factor for AD.

(Omura et al., 2022)

Frontotemporal Dementia- DSM V Criteria

A. Meets the criteria for major or mild neurocognitive disorder

B. Insidious onset and gradual progression

C. Either (1) or (2):

1. Behavioral variant:

a. Three or more of the following behavioral symptoms:

i. Behavioral disinhibition

ii. Apathy

iii. Loss of sympathy or empathy

iv. Perseverative, stereotyped or compulsive/ritualistic behavior.

v. Hyperorality and dietary changes.

b. Prominent decline in social cognition and/or executive abilities.

2. Language variant:

Prominent decline in language ability, in the form of speech production, word finding, object naming, grammar, or word comprehension.

Frontotemporal Dementia- DSM V Criteria

- D. Relative sparing of learning and memory and perceptual-motor function.
- E. The disturbance is not better explained by cerebrovascular disease, another neurodegenerative disease, the effects of a substance, or another mental, neurological, or systemic disorder.

Probable frontotemporal neurocognitive disorder is diagnosed if either of the following is present; otherwise, **possible frontotemporal neurocognitive disorder** should be diagnosed:

1. Evidence of a causative frontotemporal neurocognitive disorder genetic mutation, from either family history or genetic testing.
2. Evidence of disproportionate frontal and/or temporal lobe involvement from neuroimaging.

Epidemiology

The disease constitutes approximately 5 percent of all irreversible dementias.

Approximately 60% of cases have onset between 45-64 years, and about 30% of cases occur in individuals older than 65 years.

Prevalence estimates of behavioral variant and semantic language variant are higher among males, especially those who have a first-degree relative with the condition, and prevalence estimates of non-fluent language variant are higher among females.

(Puppala, Gorthi, Chandran, & Gundabolu, 2021)

Course And Prognosis

- The disease is gradually progressive, with median survival being 6–11 years after symptom onset and 3–4 years after diagnosis.
- Survival is shorter and decline is faster in major or mild frontotemporal NCD than in typical Alzheimer's disease.
- Presence of motor neuron disease is associated with a more rapid deterioration.

(Puppala, Gorthi, Chandran, & Gundabolu, 2021)

Differential Diagnosis

- The early stages of Pick disease/ FTD are more often characterized by personality and behavioral changes, with relative preservation of other cognitive functions, and it typically begins before 65 years of age.
- In major or mild NCD due to Alzheimer's disease, decline in learning and memory is an early feature.
- In major or mild NCD with Lewy bodies, core and suggestive features of Lewy bodies must be present.
- In major or mild NCD due to Parkinson's disease, spontaneous parkinsonism emerges well before the cognitive decline.
- In major or mild vascular NCD, depending on affected brain regions, there will be loss of executive ability and behavioral changes such as apathy.

Case Study

A 77- year- old right- handed woman is referred to clinic by her family. They report that she has become progressively more 'childish' over the past 2 years. She is inappropriately familiar with strangers and displays total disregard for etiquette such as table manners. The interviewer notice that her clothes are dirty and that she is wearing three scarves, two pairs of trousers, and two tops. Her husband, who is well kempt, reports that she insists on re- wearing dirty clothes and that he is unable to intervene. The patient tells you that there is a man living in her attic; she has never seen him but she knows that he is there. On examination, you note the presence of primitive reflexes (grasp, palmomental, pout), as well as deltoid fasciculations, thinning of the first dorsal interosseous and thenar eminence bilaterally, slow tongue movements, and hyperreflexia. The ACE- III (Addenbrooke's Cognitive Examination) score is 85/100 (attention 16/ 18, memory 21/ 26, fluency 10/ 14, language 21/ 26, visuospatial 16/ 16). Her language deficits are in naming, and you note that she has some mild semantic impairment for these items. (Puppala et al., 2021)

Causes

Genetic factors have been identified, such as mutations in the gene encoding the microtubule associated protein tau (MAPT) (40%-50% of cases), 40– 50% of cases have inclusions of TDP- 43 (43- kDa TAR DNA- binding protein) (Piguet et al., 2019) and 5% inclusions of FUS (fused in sarcoma) (Seelaar et al., 2020). These proteins are both involved in RNA (ribonucleic acid) processing, but their pathogenic mechanisms are not yet known.

The mutations in the gene encoding the microtubule associated C9ORF72 gene. This must be separately tested and is particularly suggested by the presence of motor neuron disease (DeJesus- Hernandez et al., 2021; Renton et al., 2020) or prominent psychiatric features in the proband or family members (Arighi et al., 2022; Snowden et al., 2021).

FTD is highly heritable, with up to 50% of patients having a family history of the disorder. A single causative gene can be demonstrated in approximately half of these cases, with the proportion rising in clear autosomal dominant inheritance patterns with multiple family members affected (Rohrer et al., 2019).

Dementia With Lewy Bodies (DLB)- DSM V Criteria

A. Meets the criteria for major or mild neurocognitive disorder

B. Insidious onset and gradual progression

C.

1. Core diagnostic features:

a. Fluctuation in cognition with pronounced variations in attention and alertness.

b. Recurrent visual hallucinations that are well formed and detailed.

c. Spontaneous features of parkinsonism, with onset subsequent to the development of cognitive decline.

2. Suggestive diagnostic features:

a. Meets criteria for rapid eye movement sleep behavior disorder

b. Severe neuroleptic sensitivity (e.g., drowsiness, parkinsonism, and falls).

For probable major or mild neurocognitive disorder with Lewy bodies, the individual has two core features, or one suggestive feature with one or more core features.

For possible major or mild neurocognitive disorder with Lewy bodies, the individual has only one core feature, or one or more suggestive features.

Dementia With Lewy Bodies (DLB)- DSM V Criteria

D. The disturbance is not better explained by cerebrovascular disease, another neurodegenerative disease, the effects of a substance, or another mental, neurological, or systemic disorder.

Associated Features:

- Spontaneous motor features of parkinsonism (postural instability , gait difficulty, and tremor) but only in 20% of cases
- Repeated falls
- Transient episodes of unexplained loss of consciousness
- Autonomic dysfunction, such as orthostatic hypotension and urinary incontinence,
- Auditory and other nonvisual hallucinations
- Systematized delusions, delusional misidentification,
- Depression

Lewy Body Dementia (LBD)

There are several possible symptoms of LBD, which can be grouped into the following categories:

Movement issues



- Slowed movements.
- Rigidity or stiffness.
- Tremors.
- Balance problems.
- Shuffling walk.
- Difficulty swallowing.
- Frequent falls.
- Loss of coordination.

Fluctuating cognitive function



- Visual hallucinations.
- A decline in:
- Planning abilities.
 - Problem-solving skills.
 - Decision-making
 - Memory.
 - Ability to focus.
 - Understanding visual information.

Sleep problems



- Rapid eye movement (REM) sleep behavior disorder (RBD).
- Excessive daytime drowsiness.
- Changes in sleep patterns.
- Insomnia.

Mood and behavior changes



- Depression.
- Anxiety.
- Agitation, restlessness or aggression.
- Delusions.
- Paranoia

Dysautonomia: autonomic nervous system (ANS) that doesn't function as it should



- Changes in body temperature.
- Blood pressure fluctuations.
- Dizziness.
- Fainting.
- Sensitivity to heat and cold.
- Sexual dysfunction.
- Urinary incontinence.
- Fecal (bowel) incontinence.
- Constipation.

Epidemiology

In population-based clinical studies, prevalences of around 0.7 per cent for DLB in the 65+ age group have been reported suggesting that it could account for up to 10 per cent of all dementia cases.

A recent community study of 85+ year olds found 3.3 per cent probable, 1.7 per cent possible representing 22 per cent of all demented cases.

(Holmes et al., 2019; Rahkonen et al., 2021)

Course And Prognosis

- The cognitive decline is manifested early in the course of illness, at least a year before the onset of motor symptoms.
- Disease course may be characterized by occasional plateaus but eventually progresses through severe dementia to death.
- Average duration of survival is 5–7 years with onset of symptoms when an individual is in their mid-70s.

(Holmes et al., 2019; Rahkonen et al., 2021)

Differential Diagnosis

- For NCD due to Parkinson's disease, the individual must develop cognitive decline in the context of established Parkinson's disease; by convention, the decline should not reach the stage of major NCD until at least 1 year after Parkinson's is diagnosed.
- If less than a year has passed since the onset of motor symptoms but after the onset of cognitive decline, the diagnosis is NCDLB.
- In patients with a prior diagnosis of Parkinson's disease, the onset of visual hallucinations and fluctuating cognitive impairment may be attributed to side-effects of antiparkinsonian medications, and this must be tested by dose reduction or withdrawal.

Case Study

A 73- year- old woman is referred by her family doctor because of a 3-year history of hallucinations. These occur when she is tired or the lighting is poor. The commonest manifestation is that she will make three or four coffees for people sitting on the sofa and her husband will have to remind her that it is only the two of them in the house. She also frequently hallucinates individuals in the garden and, on one occasion, thought that they were stealing her furniture. She has good retrospective insight into the fact that these people are not real but finds it very confusing at the time. Her cognition is fluctuant, with good days and bad days. She has REM sleep behaviour disorder. On examination, she has a mild parkinsonian motor syndrome and her sense of smell is poor. Despite only scoring 53/ 100 on the ACE- III (attention 11/ 18, memory 12/ 26, fluency 7/ 14, language 14/ 26, visuospatial 9/ 16), she is still able to travel to and from town alone on the bus once a week where she follows a fixed routine of going to Marks and Spencer's and coming home again. Her MRI scan demonstrated mild global atrophy with preserved hippocampal volume.

Causes

- Lewy bodies are commonly seen in familial cases of Alzheimer's disease.
- There are recent reports that triplication of the alpha-synuclein gene (SNCA) may cause DLB as its mutation can be seen in DLB, Parkinson's disease, and Parkinson's disease dementia.
- *APOE* (apolipoprotein E) is already known to increase the risk of developing Alzheimer's disease. There's growing evidence that it also increases the risk for dementia with Lewy bodies.
- Age is considered the greatest risk factor. No specific lifestyle factor has been proven to increase one's risk for DLB.
- Other known risk factors for DLB include certain diseases and health conditions, particularly Parkinson's disease and REM sleep behavior disorder, which have been linked to a higher risk of DLB.

Vascular Dementia- DSM V Criteria

A. Meets the criteria for major or mild neurocognitive disorder

B. The clinical features are consistent with a vascular etiology:

1. Onset of the cognitive deficits is temporally related to one or more cerebrovascular events.
2. Evidence for decline is prominent in complex attention (including processing speed) and frontal-executive function.

C. Evidence of cerebrovascular disease from history, physical examination and neuroimaging which is the cause for neurocognitive deficits

D. The symptoms are not better explained by another brain disease or systemic disorder.

For probable vascular neurocognitive disorder if one of the following is present:

1. Neuroimaging evidence of significant parenchymal injury attributed to cerebrovascular disease.
2. The neurocognitive syndrome is temporally related to one or more documented cerebrovascular events.
3. Both clinical and genetic evidence of cerebrovascular disease is present.

Vascular Dementia- DSM V Criteria

Possible vascular neurocognitive disorder is diagnosed if the clinical criteria are met but neuroimaging is not available and the temporal relationship of the neurocognitive syndrome with one or more cerebrovascular events is not established.

Associated Features:

- H/O of stroke and/or transient ischemic episodes
- Brain infarctions
- Personality and mood changes
- Abulia (inability to act decisively)
- Emotional lability
- Depression

Among older adults late-onset of depressive symptoms along with psychomotor slowing and executive dysfunction with progressive small vessel ischemic disease

Epidemiology

- Major or mild vascular NCD is the second most common cause of NCD after Alzheimer's disease.
- Vascular dementia (VaD) is the second leading cause of late-life dementia and accounts for about 30% of dementia in LMICs including India.
- In neuropathology series, the prevalence of vascular dementia increases from 13% at age 70 years to 44.6% at age 90 years or older and it is higher in males than females.
- Estimates are complicated, however, by the prevalence of mixed dementias, often vascular dementia in combination with Alzheimer disease pathology, with considerably less than 15% of cases arising from “pure” vascular pathologies in neuropathological studies.

(Arshad et al., 2022; Forrest & Kovacs, 2023)

Course And Prognosis

- Vascular dementia tends to progress over time. Treatments can't cure the disease, but lifestyle changes and medicines to treat underlying causes (such as high blood pressure, high cholesterol, diabetes, or blood clots) might help slow its progress.
- A person with vascular dementia may eventually need full-time nursing care or to stay in a long-term care facility.

(Arshad et al., 2022; Forrest & Kovacs, 2023)

Differential Diagnosis

Recent reports in the neuropathological and neuropsychological literature posit that **vascular dementia and Alzheimer disease** frequently co-occur, making so-called pure cases of either condition comparatively rare. If there is history of memory deficit early in the course, and progressive worsening of memory, language, executive function, and perceptual-motor abilities in the absence of corresponding focal lesions on brain imaging, are suggestive of **Alzheimer's disease as the primary diagnosis**.

NCD with Lewy bodies is distinguished from **major or mild vascular NCD** by its core features of fluctuating cognition, visual hallucinations, and spontaneous parkinsonism. While deficits in executive function and language occur in major or mild vascular NCD, the insidious onset and gradual progression of behavioral features or language impairment are characteristic of **frontotemporal NCD** and are not typical of vascular etiology.

Other medical conditions. A diagnosis of major or mild vascular NCD is not made if other diseases (e.g., brain tumor, multiple sclerosis, encephalitis, toxic or metabolic disorders) are present.

Case Study

The patient, a right-handed male, 53 years old, with a college education, had a transient ischemic attack (TIA) in 2011, and was treated in the Department of Vascular Surgery and Endovascular Procedures at the John Paul II Hospital, in Krakow, Poland. During the attack, which lasted for a few minutes, he was confused and had trouble speaking, trouble seeing in both eyes, difficulty walking, problems with balance and coordination, and odd behaviour. All symptoms resolved after a few hours. A year later, the patient was referred to the neurology department because of double vision, which resolved after two weeks of treatment. But patient complained about the deterioration of attention and memory, difficulty to make decisions, feeling of sadness, and always ready to explode or bounce back at family members which was not there before.

(Supinska et al., 2020)

Causes

The pathogenesis of vascular dementia is understood as follows: vascular risk factors lead to cerebrovascular disease that, in turn, causes brain injury; the resulting disruption of cognitive networks culminates in vascular dementia.

CT and magnetic resonance imaging (**MRI**) studies on vascular dementia have shown that infarcts associated with vascular dementia tend to be bilateral, multiple (more than two), and located in the dominant hemisphere and in the limbic structures (frontolimbic or prefrontal– subcortical and medial–limbic or medial–hippocampal circuits).

(Bir et al., 2021; Iadecola, 2013)

Risk Factors

Risk factors for vascular dementia can be divided into **vascular factors** (e.g. arterial hypertension, atrial fibrillation, myocardial infarction, coronary heart disease, diabetes, generalized atherosclerosis, lipid abnormalities, smoking), **demographic factors** (e.g. age, education), **genetic factors** (e.g. family history), and **stroke-related factors** (e.g. type of cerebrovascular disease, site and size of stroke).

Hypoxic ischaemic events (cardiac arrhythmias, congestive heart failure, myocardial infarction, seizures, pneumonia) may be an important risk factor for incident dementia in patients with stroke.

(Bir et al., 2021; Iadecola, 2013)

Assessment Tools

Screening Tool	Domain	Clinical utility
Mini- Mental State Examination (MMSE)	Attention, memory, language (brief), visuospatial ability	Brief administration time, but especially weighted towards attention and memory,
Addenbrooke's Cognitive Examination (ACE- III)	Attention, memory, executive abilities, language, visuospatial ability	comprehensive assessment of language. Helpful breakdown of tasks into subtasks of specific cognitive domains
Digit Span Test	Attention and concentration, working memory	Impaired in delirium; also impaired in AD
Trail Making Test	Speed of information processing (Part A); executive abilities (set- shifting; Part B)	Common test of executive impairment
Neuropsychiatric Inventory (NPI), Cambridge Behavioural Inventory (CBI), Frontal Behavioral Inventory (FBI)	Behavioural disturbance and functional capacity	Useful in screening for, quantitating, and characterizing behavioural deficits; especially useful in FTD

Management

- Early diagnosis is a prerequisite for adequate treatment and care
- **Non-pharmacological management** would depend upon nature of the patients' changed behaviour and current problems (e.g., behavioral therapy):
 - 1) A well-structured programme for daily activities considering the patients premorbid personality and interests, 'routinizing therapy'.
 - 2) **Reminiscence therapy** might include things like talking with your loved one about their hometown, school days, work life, or favorite hobbies. It can be done one-on-one or in groups as part of an organized therapy.
 - 3) **Family Counselling** (e.g., psycho-education, supportive psychotherapy, breaking the bad news integrating with grief therapy)
 - 4) **Cognitive stimulation therapy (CST)** is a structured program for groups of people with mild to moderate dementia. At meetings, the group does mentally engaging activities, like talking about current events, singing, playing word games, or cooking from a recipe.
 - 5) **Respite care** through placement in long-term rehabilitative homes.